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NEWS 4	AUG 24	ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced
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NEWS 6	SEP 09	50 Millionth Unique Chemical Substance Recorded in CAS REGISTRY
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NEWS 12	DEC 01	FRFULL Content and Search Enhancements
NEWS 13	DEC 01	DGENE, USGENE, and PCTGEN: new percent identity feature for sorting BLAST answer sets
NEWS 14	DEC 02	Derwent World Patent Index: Japanese FI-TERM thesaurus added
NEWS 15	DEC 02	PCTGEN enhanced with patent family and legal status display data from INPADOCDB
NEWS 16	DEC 02	USGENE: Enhanced coverage of bibliographic and sequence information
NEWS 17	DEC 21	New Indicator Identifies Multiple Basic Patent Records Containing Equivalent Chemical Indexing in CA/CAplus
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=> s calcitonin gene related peptide or CGRP
L1       31928 CALCITONIN GENE RELATED PEPTIDE OR CGRP

=> s pituitary adenylate cyclase activating peptide or PACAP
L2        9041 PITUITARY ADENYLATE CYCLASE ACTIVATING PEPTIDE OR PACAP

=> s l1 or l2
L3       40576 L1 OR L2

=> s l3 and interstitial cystitis
L4          43 L3 AND INTERSTITIAL CYSTITIS

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5           27 DUP REM L4 (16 DUPLICATES REMOVED)
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=> d bib abs 1-
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L5 ANSWER 1 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 1
AN 2009:24949 BIOSIS
DN PREV200900024949
TI Functional and Immunohistochemical Characterization of CB1 and CB2
Receptors in Rat Bladder.
AU Hayn, Matthew H. [Reprint Author]; Ballesteros, Inmaculada; de Miguel,
Fernando; Coyle, Christian H.; Tyagi, Shachi; Yoshimura, Naoki;
Chancellor, Michael B.; Tyagi, Pradeep
CS Univ Pittsburgh, Med Ctr, Dept Urol, Kaufmann Bldg,Suite 700,3451 5th Ave,
Pittsburgh, PA 15213 USA
haynm2@upmc.edu
SO Urology, (NOV 2008) Vol. 72, No. 5, pp. 1174-1178.
ISSN: 0090-4295.
DT Article
LA English
ED Entered STN: 17 Dec 2008
Last Updated on STN: 17 Dec 2008
AB OBJECTIVES To determined the localization of CB1 and CB2 receptors in rat
bladder and investigate the effect of a mixed CB1/CB2 receptor agonist,
ajulemic acid (AJA), on chemically evoked release of the sensory
neuropeptide calcitonin gene-retated peptide (CGRP).METHODS
Whole rat bladders were incubated in a series of tissue baths containing
physiologic salt solution to measure baseline CGRP release by
enzyme immunoassay. Capsaicin (30 nM) and adenosine triphosphate (10 mu
M) were used to provoke CGRP release in the presence or absence
of AJA. Specificity of AJA for CB1 and CB2 receptors was determined using
antagonists. Localization was determined by immunofluorescence for CB1
and CB2 receptors in fixed bladders.RESULTS Immunofluorescence, showed the
localization of CB1 and CB2 receptors in the bladder. Mean baseline
CGRP release was 605 +/- 62 pg/g of bladder weight, and AJA had no
effect on CGRP release. The addition of adenosine
triphosphate/capsaicin significantly increased the CGRP release
over baseline, by 44% (P < .05), and AJA application significantly

decreased CGRP release, by 29% compared with controls (P < .05). The CB1 and CB2 antagonists AM 251 and AM 630, respectively, reversed the blunting effect of AJA on evoked CGRP release, resulting in an increase of 40% and 38% over baseline, respectively.CONCLUSIONS CB1 and CB2 receptors are localized in the urothelium of rat bladder, and application of AJA inhibits the evoked release of CGRP by acting on CB1 and CB2 receptors. These findings identify a potential new pathway for study in the evaluation and treatment of painful bladder syndrome/ interstitial cystitis. UROLOGY 72: 1174-1178, 2008. (C) 2008 Elsevier Inc.

L5 ANSWER 2 OF 27 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2008366272 EMBASE
TI Reply.
AU Liu, Hsin-Tzu, Dr. (correspondence); Kuo, Hann-Chorng
CS Department of Urology, Buddhist Tzu Chi General Hospital, Tzu Chi University, Hualien, Taiwan, Province of China.
SO Urology, (August 2008) Vol. 72, No. 2, pp. 464.
Refs: 5
ISSN: 0090-4295; E-ISSN: 1527-9995 CODEN: URGYAZ
PB Elsevier Inc., 360 Park Avenue South, New York, NY 10010, United States.
PUI S 0090-4295(08)00228-8
CY United States
DT Journal; Letter
FS 028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LA English
ED Entered STN: 4 Sep 2008
Last Updated on STN: 4 Sep 2008

L5 ANSWER 3 OF 27 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2008366271 EMBASE
TI Intravesical Botulinum Toxin A Injections Plus Hydrodistension Can Reduce Nerve Growth Factor Production and Control Bladder Pain in Interstitial Cystitis: A Molecular Mechanism.
AU Namazi, Hamid, Dr. (correspondence)
CS Department of Orthopaedic Surgery, Shiraz University of Medical Sciences, Chamran Hospital, Shiraz, Iran, Islamic Republic of.
SO Urology, (August 2008) Vol. 72, No. 2, pp. 463-464.
Refs: 6

ISSN: 0090-4295; E-ISSN: 1527-9995 CODEN: URGYAZ
PB Elsevier Inc., 360 Park Avenue South, New York, NY 10010, United States.
PUI S 0090-4295(08)00227-6
CY United States
DT Journal; Letter
FS 028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LA English
ED Entered STN: 4 Sep 2008
Last Updated on STN: 4 Sep 2008

L5 ANSWER 4 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 2
AN 2008:134998 BIOSIS
DN PREV200800124454
TI Botulinum toxin type A inhibits sensory neuropeptide release in rat
bladder models of acute injury and chronic inflammation.
AU Lucioni, Alvaro [Reprint Author]; Bales, Gregory T.; Lotan, Tamara L.;
McGehee, Daniel S.; Cook, Sean P.; Rapp, David E.
CS Univ Chicago, Pritzker Sch Med, Dept Surg, Urol Sect, 5841S,Maryland
Ave,MC 6038, Chicago, IL 60637 USA
alvarolucioni@hotmail.com
SO BJU International, (FEB 2008) Vol. 101, No. 3, pp. 366-370.
ISSN: 1464-4096.
DT Article
LA English
ED Entered STN: 20 Feb 2008
Last Updated on STN: 20 Feb 2008
AB To determine the effect of botulinum toxin type A (BTX-A) on the release
of the neuropeptides substance P (SP) and calcitonin
gene-related peptide (CGRP) from
isolated bladder preparations after acute injury with HCl and the
induction of cyclophosphamide (CYP)-induced cystitis, as neurogenic
inflammation has been increasingly identified in urological disorders such
as interstitial cystitis.Adult rats had either an
intraperitoneal injection with CYP or saline over a 10-day period to
induce chronic bladder inflammation, after which the bladder was
harvested, or normal bladder explants were injured acutely with incubation
(20 s) in HCl (0.4 M). To measure the effect of BTX-A on the release of
neurotransmitters, harvested bladders were incubated in an organ bath

containing BTX-A (10 U) or vehicle. Bladders were transferred to a subsequent bath (physiological saline) and incubated for 15 min, and the bathing medium analysed to measure neurotransmitter release, as determined by radioimmunoassay. Bladder specimens from sham treatment, controls and experimental rats were compared histologically.Acute injury with HCl caused a significantly greater release of both CGRP and SP release (1235 and 1655 pg/g, respectively) than in controls (183 and 449 pg/g, respectively; P < 0.001). This increase in neurotransmitter release was partly inhibited by exposure to BTX-A (870 and 1033 pg/g (P < 0.05 and < 0.01). CYP-induced chronic inflammation caused significantly greater release of SP than in the controls (1060 and 605 pg/g, respectively; P < 0.005). Exposure to BTX-A partly inhibited the release of SP after CYP-induced cystitis (709 pg/g, P < 0.05).The application of BTX-A inhibits the release of sensory neurotransmitters from isolated bladder preparations in rat bladder models of both acute injury and chronic inflammation, suggesting a potential clinical benefit of BTX-A in the treatment of neurogenic inflammation.

L5 ANSWER 5 OF 27 EMBASE
AN 2008555557 EMBASE
TI Inside information: The unique features of visceral sensation.
AU Robinson, David R.; Gebhart, G.F.
CS Department of Anesthesiology, Pittsburgh Center for Pain Research, University of Pittsburgh, Pittsburgh, PA 15213, United States.
AU Robinson, D. R., Dr. (correspondence)
CS Department of Anesthesiology, Pittsburgh Center for Pain Research, University of Pittsburgh, Pittsburgh, PA 15213, United States.
SO Molecular Interventions, (1 Oct 2008) Vol. 8, No. 5, pp. 242-253.
Refs: 76
ISSN: 1534-0384; E-ISSN: 1543-2548 CODEN: MIONAR
PB American Society for Pharmacology and Experimental Therapy, 9650 Rockville

Pike, Bethesda, MD 20814, United States.
CY United States
DT Journal; General Review; (Review)
FS 002 Physiology
008 Neurology and Neurosurgery
048 Gastroenterology
LA English
SL English
ED Entered STN: 19 Dec 2008
Last Updated on STN: 19 Dec 2008
AB Most of what is written and believed about pain and nociceptors originates
from studies of the "somatic" (non-visceral) sensory system. As a result,
the unique features of visceral pain are often overlooked. In the clinic,
the management of visceral pain is typically poor, and drugs that are used
with some efficacy to treat somatic pain often present unwanted effects on
the viscera. For these reasons, a better understanding of visceral
sensory neurons - particularly visceral nociceptors - is required. This
review provides evidence of functional, morphological, and biochemical
differences between visceral and non-visceral afferents, with a focus on
potential nociceptive roles, and also considers some of the potential
mechanisms of visceral mechanosensation.

L5 ANSWER 6 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2008:74342 BIOSIS
DN PREV200800073712
TI Repeated botulinum toxin injections: A new answer for further questions.
AU Lazzeri, Massimo [Reprint Author]
CS Dept Urol, Santa Chiara Firenze Giomi Grp, Pzza INdipendenza 11, I-50129
Florence, Italy
lazzeri.m@tiscali.it
SO European Urology, (DEC 2007) Vol. 52, No. 6, pp. 1571-1573.
CODEN: EUURAV. ISSN: 0302-2838.
DT Article
Editorial
LA English
ED Entered STN: 16 Jan 2008
Last Updated on STN: 16 Jan 2008

L5 ANSWER 7 OF 27 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights

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AN 2007494550 EMBASE
TI Resiniferatoxin and botulinum toxin type A for treatment of lower urinary
tract symptoms.
AU Cruz, Francisco, Dr. (correspondence); Dinis, Paulo
CS Department of Urology, Hospital de S. Joao, Faculty of Medicine/IBMC of
Porto, Porto, Portugal. cruzfjmr@med.up.pt
AU Cruz, Francisco, Dr. (correspondence)
CS Department of Urology, Hospital de S. Joao, P-4200 Porto, Portugal.
cruzfjmr@med.up.pt
SO Neurourology and Urodynamics, (2007) Vol. 26, No. 6 SUPPL., pp. 920-927.
Refs: 57
ISSN: 0733-2467; E-ISSN: 1520-6777 CODEN: NEUREM
CY United States
DT Journal; Conference Article; (Conference paper)
FS 028 Urology and Nephrology
037 Drug Literature Index
006 Internal Medicine
008 Neurology and Neurosurgery
LA English
SL English
ED Entered STN: 23 Oct 2007
Last Updated on STN: 23 Oct 2007
AB Resiniferatoxin (RTX) and botulinum toxin subtype A (BTX-A) are increasingly viewed as potential treatments for lower urinary tract
symptoms (LUTS) refractory to conventional therapy. RTX, a capsaicin
analogue devoid of severe pungent properties, acts by desensitizing the
transient receptor potential vanilloid type 1 (TRPV1) receptor and
inactivating C-fibers. BTX-A cleaves soluble N-ethylmaleimide-sensitive
factor attachment protein receptor (SNARE) proteins in afferent and
efferent nerve endings, therefore impeding the fusion of synaptic vesicles
with the neuronal membrane necessary for the release of neurotransmitters.
In patients with neurogenic and idiopathic detrusor overactivity, RTX and
BTX-A have been shown to increase the volume to first detrusor
contraction, increase bladder capacity, and improve urinary incontinence
and quality of life. Recent data also suggest a role for these
neurotoxins in treating urgency, the primary symptom in overactive bladder

(OAB) syndrome. Furthermore, experimental data strongly support the use of both neurotoxins in the treatment of pain and frequency in patients with interstitial cystitis/painful bladder syndrome (IC/PBS), although the results from available clinical trials for this use are still inconclusive. In spite of promising results overall, it should be made clear that the administration of these neurotoxins is still considered an experimental procedure and that more clinical studies are necessary before a license for their use will be issued by health authorities. .COPYRGT. 2007 Wiley-Liss, Inc.

L5 ANSWER 8 OF 27 EMBASE
AN 2007245559 EMBASE
TI New insights into the pathogenesis of fibromyalgia syndrome: Important role of peripheral and central pain mechanisms.
AU Staud, Roland (correspondence)
CS Division of Rheumatology and Clinical Immunology, McKnight Brain Institute, University of Florida, Gainesville, FL 32610-0221, United States. staudr@ufl.edu
AU Staud, Roland (correspondence)
CS Department of Medicine, University of Florida, College of Medicine, Gainesville, FL 32610-0221, United States. staudr@ufl.edu
SO Current Rheumatology Reviews, (May 2007) Vol. 3, No. 2, pp. 113-121.
Refs: 143
ISSN: 1573-3971
CY Netherlands
DT Journal; General Review; (Review)
FS 026 Immunology, Serology and Transplantation
003 Endocrinology
031 Arthritis and Rheumatism
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LA English
SL English
ED Entered STN: 26 Jun 2007
Last Updated on STN: 26 Jun 2007
AB Clinical symptoms of chronic muscle conditions like fibromyalgia (FM), include pain, stiffness, subjective weakness, and muscle fatigue. Pain in FM is usually described as fluctuating and always associated with local or

generalized tenderness (hyperalgesia and/or allodynia). This tenderness related to FM pain depends on increased peripheral and/or central nervous system responsiveness to peripheral stimuli which can be either noxious (hyperalgesia) or non-noxious (allodynia). For example, patients with muscle hyperalgesia will rate painful muscle stimuli higher than normal controls, whereas patients with allodynia may perceive light touch as painful, something that a "normal" individual will never describe as painful. The pathogenesis of such peripheral and/ or central nervous system changes in FM is unclear, but peripheral tissue changes, specifically in muscles have been implicated. Indirect evidence from interventions that attenuate tonic peripheral impulse input in patients with FM suggest that overall FM pain is dependent on signals from deep tissues. More importantly, allodynia and hyperalgesia can be improved or abolished by removal of peripheral impulse input. Another potential mechanism for FM pain is central disinhibition. However, this pain mechanism also depends on tonic impulse input even if only inadequately inhibited. Thus a promising approach to understanding FM pain is to determine whether abnormal activity of receptors in deep tissues is fundamental to the development and maintenance of this chronic pain disorder.

L5 ANSWER 9 OF 27 BIOSIS
AN 2007:22168 BIOSIS
DN PREV200700036860
TI PACAP enhances mouse urinary bladder contractility and is upregulated in micturition reflex pathways after cystitis.
AU Herrera, Gerald M.; Braas, Karen M.; May, Victor; Vizzard, Margaret A.
[Reprint Author]
CS Univ Vermont, Coll Med, Dept Neurol, D411 Given Bldg, Burlington, VT 05405
USA
margaret.vizzard@uvm.edu

SO Vaudry, H [Editor]; Laburthe, M [Editor]. Ann. N. Y. Acad. Sci., (2006)
pp. 330-336. Annals of the New York Academy of Sciences.
Publisher: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ, OXEN,
UK. Series: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES.
Meeting Info.: 7th International Symposium on VIP, PACAP and Related
Peptides. Rouen, FRANCE. September 11 -14, 2005. Conseil Reg
Haute-Normandie; Agglomerat Rouen; Inst Fed Rech Multidisciplinaires
Peptides; Inst Natl Sante Rech Med; Municipal Rouen; Sci Act
Haute-Normandie; Tech Chime-Biol Sante; Univ Paris 7; Univ Rouen.
CODEN: ANYAA9. ISSN: 0077-8923. ISBN: 1-57331-550-8(H).
DT Book; (Book Chapter)
Conference; (Meeting)
LA English
ED Entered STN: 27 Dec 2006
Last Updated on STN: 11 Jul 2007
AB Pituitary adenylate cyclase-activating polypeptide (PACAP)
elicits a transient contraction, sustained increase in the amplitude of
spontaneous phasic contractions, and significantly increases the amplitude
of nerve-mediated contractions in mouse urinary bladder smooth muscle
(UBSM) strips. PACAP immunoreactivity (IR) is increased in
micturition reflex pathways following cystitis. PACAP may
contribute to altered sensation and bladder overactivity in the chronic
bladder inflammatory syndrome, interstitial cystitis.

L5 ANSWER 10 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN DUPLICATE 3
AN 2006:290216 BIOSIS
DN PREV200600295152
TI Botulinum toxin type A inhibits calcitonin gene-
related peptide release from isolated rat bladder.
AU Rapp, David E. [Reprint Author]; Turk, Katherine W.; Bales, Gregory T.;
Cook, Sean P.
CS Univ Chicago, Pritzker Sch Med, Dept Surg, Urol Sect, 5841 S Maryland
Ave,MC 6038, Chicago, IL 60637 USA
derapp@yahoo.com
SO Journal of Urology, (MAR 2006) Vol. 175, No. 3, Part 1, pp. 1138-1142.
CODEN: JOURAA. ISSN: 0022-5347.
DT Article
LA English

ED Entered STN: 31 May 2006
Last Updated on STN: 31 May 2006
AB Purpose: Increasing evidence suggests that sensory nerve dysfunction may underlie several urological disorders, including interstitial cystitis and sensory urgency. We determined the effect of botulinum toxin type A (Allergan, Irvine, California) on baseline and chemically evoked release of the sensory neuropeptide, calcitonin gene-related peptide in an isolated bladder preparation.Materials and Methods: Whole rat bladders were incubated in a series of tissue baths containing physiological salt solution. Following bladder equilibration in PSS sequential incubation was performed and this sample was used to measure baseline CGRP release. To evoke CGRP release tissue was subsequently incubated in PSS containing capsaicin (30 nM) and adenosine triphosphate (10 mu M). To measure the effect of BTX-A on baseline and evoked CGRP release bladders were incubated for 6 hours in an organ bath containing BTX-A (50 mu M) or vehicle prior to bladder equilibration. CGRP release was determined by radioimmunoassay.Results: Mean baseline release of CGRP SEM was 346 44 pg/gm. Adenosine triphosphate/capsaicin application increased CGRP release by 75% over baseline (606 +/- 98 pg/gm, p < 0.005). BTX-A application resulted in a 19% decrease in baseline release of CGRP, although this difference did not achieve statistical significance. BTX-A application significantly decreased evoked CGRP by 62% vs control (606 +/- 98 vs 229 +/- 21 pg/gm, p < 0.005).Conclusions: BTX-A application inhibits the evoked release of CGRP from afferent nerve terminals in isolated rat bladder. This finding suggests a potential clinical benefit of BTX-A for the treatment of interstitial cystitis or sensory urgency.

L5 ANSWER 11 OF 27 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 4
AN 2006:983267 CAPLUS
DN 145:500253
TI PACAP enhances mouse urinary bladder contractility and is upregulated in micturition reflex pathways after cystitis
AU Herrera, Gerald M.; Braas, Karen M.; May, Victor; Vizzard, Margaret A.
CS Department of Pharmacology, University of Vermont College of Medicine, Burlington, VT, 05405, USA
SO Annals of the New York Academy of Sciences (2006), 1070(VIP, PACAP, and

Related Peptides), 330-336
CODEN: ANYAA9; ISSN: 0077-8923
PB Blackwell Publishing, Inc.
DT Journal
LA English
AB Pituitary adenylate cyclase-activating polypeptide (PACAP) elicits a transient contraction, sustained increase in the amplitude of spontaneous phasic contractions, and significantly increases the amplitude of nerve-mediated contractions in mouse urinary bladder smooth muscle (UBSM) strips. PACAP immunoreactivity (IR) is increased in micturition reflex pathways following cystitis. PACAP may contribute to altered sensation and bladder overactivity in the chronic bladder inflammatory syndrome, interstitial cystitis.
OSC.G 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)
RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 12 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2006:221849 BIOSIS
DN PREV200600225257
TI Role for pituitary adenylate cyclase activating polypeptide (PACAP) in cystitis-induced plasticity of micturition reflexes.
AU Braas, K. M. [Reprint Author]; May, V.; Zvara, P.; Nausch, B.; Kliment, J.; Dunleavy, J. D.; Nelson, M.; Vizzard, M. A.
CS Univ Vermont, Coll Med, Dept Anat and Neurobiol, Burlington, VT 05405 USA
SO Regulatory Peptides, (SEP 15 2005) Vol. 130, No. 3, pp. 157-158.
Meeting Info.: 7th International Symposium on VIP, PACAP and Related Peptides. Rouen, FRANCE. September 11 -14, 2005. Conseil Reg Haute-Normandie; Agglomerat Rouen; Inst Fed Rech Multidisciplinaires Peptides; Inst Natl Sante Rech Med; Municipal Rouen; Sci Act Haute-Normandie; Tech Chime-Biol Sante; Univ Paris 7; Univ Rouen.
CODEN: REPPDY. ISSN: 0167-0115.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 5 Apr 2006
Last Updated on STN: 5 Apr 2006

L5 ANSWER 13 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on

STN
AN 2005:447062 BIOSIS
DN PREV200510235533
TI Innervation induced by cystitis. Comparison of experimental cystitis
modelin pigs versus interstitial cystitis in humans.
AU Radziszewski, P. [Reprint Author]; Bossowska, A.; Borkowski, A.; Majewski,
M.
SO European Urology Supplements, (MAR 2005) Vol. 4, No. 3, pp. 58.
Meeting Info.: 20th Annual Meeting of the European-Association-of-Urology.
Istanbul, TURKEY. 20050317,. European Assoc Urol.
ISSN: 1569-9056.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 3 Nov 2005
Last Updated on STN: 3 Nov 2005

L5 ANSWER 14 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN DUPLICATE 5
AN 2005:8558 BIOSIS
DN PREV200500004621
TI Intravesical botulinum toxin A administration produces analgesia against
acetic acid induced bladder pain responses in rats.
AU Chuang, Yao-Chi [Reprint Author]; Yoshimura, Naoki; Huang, Chao-Cheng;
Chiang, Po-Hui; Chancellor, Michael B.
CS Suite 700,Kaufmann Bldg,3471 5th Ave, Pittsburgh, PA, 15213, USA
chancellormb@msx.upmc.edu
SO Journal of Urology, (October 2004) Vol. 172, No. 4, Part 1, pp. 1529-1532.
print.
CODEN: JOURAA. ISSN: 0022-5347.
DT Article
LA English
ED Entered STN: 16 Dec 2004
Last Updated on STN: 16 Dec 2004
AB Purpose: There is evidence that botulinum toxin A (BTX-A) might have
analgesic properties. However, the mechanisms by which BTX-A alters pain
remain largely unexplored. In the bladder afferent nerve fibers contain
calcitonin gene-related peptide (
CGRP). In this study we investigated the effect of intravesical
BTX-A administration on CGRP immunoreactivity and bladder
hyperactivity in an acetic acid induced bladder pain model in rats.

Materials and Methods: Experimental and control animals were catheterized and intravesically exposed to protamine sulfate (1 ml, 10 mg/ml), followed by BTX-A (1 ml, 25 U/ml) or saline, respectively. Three or 7 days after intravesical therapy continuous cystometrograms were performed using urethane anesthesia by filling the bladder (0.08 ml per minute) with saline, followed by 0.3% acetic acid. Bladder immunohistochemistry was used to detect CGRP. Results: The intercontraction interval (ICI) was decreased after acetic acid instillation (50.2% and 65.0%) in the control group at days 3 and 7, respectively. However, rats that received BTX-A showed a significantly decreased response (28.6% ICI decrease) to acetic acid instillation at day 7. This effect was not observed at day 3 (62.2% ICI decrease). Increased CGRP immunoreactivity was detected in the BTX treated group at day 7, which was not detected at day 3. Conclusions: Intravesical BTX administration blocked acetic acid induced bladder pain responses and inhibited CGRP release from afferent nerve terminals. These results support the clinical application of BTX-A for the treatment of interstitial cystitis and other types of visceral pain.

AN 2004315260 EMBASE

TI Experimental neurogenic cystitis.

AU Jasmin, Luc (correspondence); Janni, Gabriella

CS Department of Neurological Surgery, University of California, San Francisco, CA, United States.

SO Advances in Experimental Medicine and Biology, (2004) Vol. 539 A, pp. 319-335.
Refs: 86
ISSN: 0065-2598 CODEN: AEMBAP

CY United States

DT Journal; Conference Article; (Conference paper)

FS 021 Developmental Biology and Teratology
028 Urology and Nephrology
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
009 Surgery

LA English
SL English
ED Entered STN: 12 Aug 2004
Last Updated on STN: 12 Aug 2004
AB Recent advances in basic and clinical research indicate that interstitial cystitis (IC) is a form of neurogenic inflammation, thereby opening new avenues for research into this painful disease. With this in mind, we have recently developed a rat model of neurogenic inflammation of the bladder produced by a central nervous system viral disease. As in IC, the inflammation in this model develops without direct injury or trauma to the bladder, is non-infectious, and is limited to the bladder. Our most recent studies aimed at further testing the similarity of this animal model to IC by assessing the urine content in histamine with the occurrence of mast cell degranulation in the bladder wall. We further verified for a sex difference in the occurrence of the disease. Our results showed increased levels of urine histamine and mast cell activation during the early stages of the disease. We additionally observed that females had a greater degree of plasma extravasation, while males had a greater cellular infiltration together with worse behavioral signs. Gonadectomy prevented the bladder inflammation altogether in both males and females. These findings further validate our model of neurogenic cystitis to study the neurogenic component of IC.

L5 ANSWER 16 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2004:256199 BIOSIS
DN PREV200400256219
TI Efficacy and safety of recombinant human anti-NGF antibody in the treatment of IC.
AU Dimitrakov, Jordan D. [Reprint Author]; Dikov, Dorian [Reprint Author]
CS Plovdiv, Bulgaria
SO Journal of Urology, (April 2004) Vol. 171, No. 4 Supplement, pp. 95.
print.
Meeting Info.: Annual Meeting of the American Urological Association. San

Francisco, CA, USA. May 08-13, 2004. American Urological Association.
CODEN: JOURAA. ISSN: 0022-5347.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 12 May 2004
Last Updated on STN: 12 May 2004

L5 ANSWER 17 OF 27 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights
reserved on STN
AN 2003501773 EMBASE
TI Special Contribution 1: The basics behind bladder pain: A review of data
on lower urinary tract sensations.
AU Wyndaele, J.J., Dr. (correspondence); De Wachter, Stefan
CS Department of Urology, Faculty of Medicine, University of Antwerpen,
Belgium. Jean-Jacques.Wyndaele@uza.be
AU Wyndaele, J.J., Dr. (correspondence)
CS Department of Urology, UZA, 10 Wilrijkstraat, B 2650 Edegem, Belgium.
Jean-Jacques.Wyndaele@uza.be
SO International Journal of Urology, (Oct 2003) Vol. 10, No. SUPPL., pp.
S49-S55.
Refs: 86
ISSN: 0919-8172 CODEN: IJURF3
CY Australia
DT Journal; Conference Article; (Conference paper)
FS 028 Urology and Nephrology
006 Internal Medicine
LA English
SL English
ED Entered STN: 30 Dec 2003
Last Updated on STN: 30 Dec 2003
AB Interstitial cystitis is a syndrome consisting of
frequency, urgency, and bladder pain that increases with bladder filling
and improves temporarily after voiding. The exact cause or causes are not
as yet fully understood. This leads to uncertainty in diagnosis and
treatment. There is need for more knowledge, and to acquire this for more
research. The fact that the condition causes pain, a pathologic
stimulation of sensory fibres, makes understanding the basic sensory
mechanisms in the lower urinary tract in normal and pathologic conditions
mandatory. In this article we review the data on bladder sensation from

the last 25 years and the possible relation with painful bladder syndrome.

L5 ANSWER 18 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN
AN 2002:2909 BIOSIS
DN PREV200200002909
TI Alterations in bladder afferent neurons and urothelium in cats with
interstitial cystitis.
AU Buffington, C. A. [Reprint author]; Kiss, S.; Roppolo, J. R.; de Groat, W.
C.; Dineley, K. E.; Reynolds, I. J.; Birder, L. A.
CS College Veterinary Medicine, Ohio State University, Columbus, OH, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 2163.
print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San
Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 28 Dec 2001
Last Updated on STN: 25 Feb 2002
AB Experiments were conducted in cats with feline interstitial
cystitis (IC) to evaluate whether the chemical properties and/or
intracellular signaling mechanisms in afferent neurons and epithelial
cells in the urinary bladder (UB) are altered in IC. UB dorsal root
ganglion (DRG) cells were identified by axonal tracing (fast blue) and
sections of DRG, spinal cord (SC) and UB were processed for neuropeptides
(CGRP, VIP). In IC cats, the number of CGRP
-immunoreactive dye-labelled, bladder DRG cells was increased by 50% and
the mean size of labelled DRG cells was increased (45%). Afferent (VIP,
CGRP) fiber density in UB and sacral spinal cord also increased in
IC. In addition, epithelial cells in IC cats exhibited abnormal calcium
signaling. In urothelial cells from normal cat UB, ATP mobilized
intracellular calcium via activation of P2Y receptors, whereas both P2X
and P2Y receptors were involved in this response in cells from IC cats.

In addition, compared to normal cats, cultured urothelial cells from IC cats exhibited a significant increase (90%) in stretch-evoked ATP release induced by a hypo-osmotic stimulus measured using a luciferin-luciferase assay. ATP release was blocked by gadolinium, an inhibitor of stretch activated channels. These studies revealed that IC cats have an altered urothelium which in turn may influence afferent excitability. Changes in neural-epithelial interactions may correlate with abnormal sensations in IC.

L5 ANSWER 19 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 6
AN 2001:345507 BIOSIS
DN PREV200100345507
TI Cell relationship in a wistar rat model of spontaneous prostatitis.
AU Keith, Ingegerd M. [Reprint author]; Jin, Jie; Neal, Durwood, Jr.; Teunissen, Brian D.; Moon, Timothy D.
CS Departments of Comparative Bioscience and Surgery, University of Wisconsin and Veterans Affairs Medical Center, Madison, WI, USA
SO Journal of Urology, (July, 2001) Vol. 166, No. 1, pp. 323-328. print.
CODEN: JOURAA. ISSN: 0022-5347.
DT Article
LA English
ED Entered STN: 25 Jul 2001
Last Updated on STN: 19 Feb 2002
AB Purpose: Prostatitis in men is a painful, noninfectious inflammatory condition. It is similar to interstitial cystitis which is associated with increased bladder mast cell and sensory nerve fiber density as well as suprapubic pain. Certain strains of rats may provide a useful model for studies of the development of spontaneous prostatitis. We evaluated the time course, and involvement of mast cells and sensory nerve fibers in this process using Wistar rats. Materials and Methods: The prostates of 4, 6, 8, 10 and 13-week-old male Wistar rats were examined for the degree of inflammation, innervation, mast cell

density and nerve mast cell relationship using histochemical and immunocytochemical studies. Bacterial cultures of tissue were performed at 13 weeks. Results: The inflammatory cell index increased progressively with age. Inflammation was moderate and consisted mostly of lymphocytes and macrophages associated with occasional glandular epithelial necrosis and edema. The density of nerve fibers immunoreacting with the neuronal marker protein gene produce 9.5 increased gradually with age and fibers immunopositive for the sensory neuropeptide calcitonin gene-related peptide more than doubled by 13 weeks compared with by 4 weeks. The density of visible mast cells declined after 4 weeks in a pattern that corresponded with the increased percent of mast cells undergoing degranulation. For the mast cells with calcitonin gene-related peptide immuno-positive nerve fibers within a distance of 40 mum. distance correlated significantly with the degree of degranulation. Bacterial cultures were negative at 13 weeks. Conclusions: Our results confirm previous reports of spontaneous prostatitis in Wistar rats and indicate that moderate inflammation may occur in 80% of rats at as early as age 13 weeks. While the correlation of the nerve mast cell axis with mast cell degranulation does not prove our hypothesis of mast cell mediated inflammatory mediator release in the development of nonbacterial prostatitis, it suggests that such a relationship is possible.

L5 ANSWER 20 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 7
AN 2001:284434 BIOSIS
DN PREV200100284434
TI Alterations in neuropeptide expression in lumbosacral bladder pathways following chronic cystitis.
AU Vizzard, Margaret A. [Reprint author]
CS Department of Anatomy and Neurology, University of Vermont College of Medicine, E219 Given Building, Burlington, VT, 05405, USA mvizzard@zoo.uvm.edu
SO Journal of Chemical Neuroanatomy, (March, 2001) Vol. 21, No. 2, pp.

125-138. print.
CODEN: JCNAEE. ISSN: 0891-0618.
DT Article
LA English
ED Entered STN: 13 Jun 2001
Last Updated on STN: 19 Feb 2002
AB These studies examined changes in the expression of calcitonin gene-related peptide (CGRP) and substance P (SP) in lumbosacral (L6-S1) micturition reflex pathways, following chronic cystitis induced by cyclophosphamide (CYP). In control Wistar rats, CGRP- or SP-immunoreactivity (IR) was expressed in fibers in the superficial dorsal horn in all segmental levels examined (L4-S1). Bladder afferent cells in the dorsal root ganglia (DRG; L6, S1) from control animals also exhibited CGRP-(41-55%) or SP-IR (2-3%). Following chronic, CYP-induced cystitis, CGRP- and SP-IR were dramatically increased in spinal segments and DRG (L6, S1) involved in micturition reflexes. The density of CGRP- and SP-IR was increased in the superficial laminae (I-II) of the L6 and S1 spinal segments. No changes in CGRP- or SP-IR were observed in the L4-L5 segments. Staining was also dramatically increased in a fiber bundle extending ventrally from Lissauer's tract in lamina I along the lateral edge of the DH to the sacral parasympathetic nucleus in the L6-S1 spinal segments. Following chronic cystitis, CGRP- and SP-IR in cells in the L6 and S1 DRG significantly (P ltoreq 0.05) increased and the percentage of bladder afferent cells expressing CGRP- (76%) or SP-IR (11-18%) also significantly (P ltoreq 0.001) increased. No changes were observed in the L4-L5 DRG. These studies suggest that the neuropeptides, CGRP and SP, may play a role in urinary bladder afferent pathways, following chronic urinary bladder inflammation. Changes in CGRP or SP expression following cystitis may contribute to the altered visceral sensation (allodynia) and/or urinary bladder hyperreflexia in the clinical syndrome, interstitial cystitis.

L5 ANSWER 21 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 8
AN 2000:172201 BIOSIS
DN PREV200000172201

TI Increased tyrosine hydroxylase immunoreactivity in bladder tissue from patients with classic and nonulcer interstitial cystitis .

AU Peeker, Ralph [Reprint author]; Aldenborg, Frank; Dahlstrom, Annica; Johansson, Sonny L.; Li, Jia-Yi; Fall, Magnus
CS Urology Division, Department of Surgery, Sahlgrenska University Hospital, Goteborg, Sweden

SO Journal of Urology, (April, 2000) Vol. 163, No. 4, pp. 1112-1115. print.
CODEN: JOURAA. ISSN: 0022-5347.
DT Article
LA English
ED Entered STN: 3 May 2000
Last Updated on STN: 4 Jan 2002

AB Purpose: Interstitial cystitis is a chronic debilitating condition which mainly affects women. Accumulated evidence indicates that interstitial cystitis is a heterogeneous syndrome. The nonulcer subtype appears different than classic interstitial cystitis in regard to symptoms, and endoscopic and histological findings as well as response to various treatments. We further explore the neurogenic nature of this disease using indirect immunofluorescence to evaluate the presence and density of various autonomic and sensory nerve fibers. Materials and Methods: Specimens from the bladder wall of 6 patients with classic interstitial cystitis, 7 with nonulcer interstitial cystitis and 6 controls were evaluated to determine the presence and density of nerve fibers containing tyrosine hydroxylase, calcitonin gene-related peptide, neuropeptide Y and substance P using specific antibodies, and the general presence of nerve fibers using a mixture of antibodies against nerve filament, neuron specific enolase and S-100 protein. Results: Increased density and number of nerve fibers immunoreactive for tyrosine hydroxylase were noted in interstitial cystitis cases compared to controls. Furthermore, there was a difference between classic and nonulcer disease in the overall density of nerves using the antibody mixture. Conclusions: Our findings indicate an altered

peripheral sympathetic innervation in interstitial cystitis cases, which may be an indication of primary neurogenic etiology. The difference in nerve density observed after incubation with the antibody mixture between classic and nonulcer interstitial cystitis supports the hypothesis that the 2 forms represent separate entities.

L5 ANSWER 22 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 9
AN 2000:221490 BIOSIS
DN PREV200000221490
TI Up-regulation of pituitary adenylate cyclase-activating polypeptide in urinary bladder pathways after chronic cystitis.
AU Vizzard, Margaret A. [Reprint author]
CS College of Medicine, Department of Neurology, University of Vermont, E219 Given Building, Burlington, VT, 05405, USA
SO Journal of Comparative Neurology, (May 8, 2000) Vol. 420, No. 3, pp. 335-348. print.
CODEN: JCNEAM. ISSN: 0021-9967.
DT Article
LA English
ED Entered STN: 31 May 2000
Last Updated on STN: 5 Jan 2002
AB These studies examined changes in the expression of pituitary adenylate cyclase-activating polypeptide (PACAP) in micturition reflex pathways after chronic cystitis induced by cyclophosphamide (CYP). In control Wistar rats, PACAP immunoreactivity was expressed in fibers in the superficial dorsal horn at all segmental levels examined (L1, L2, and L4-S1). Bladder afferent cells (40-45%) in the dorsal root ganglia (DRG; L1, L2, L6, and S1) from control animals also exhibited PACAP immunoreactivity. After chronic, CYP-induced cystitis, PACAP immunoreactivity increased dramatically in spinal segments and DRG (L1, L2, L6, and S1) involved in micturition reflexes. The density of PACAP immunoreactivity was increased in the superficial laminae (I-II) of the L1, L2, L6, and S1 spinal segments. No changes in PACAP immunoreactivity were observed in the L4-L5 segments. Staining also increased dramatically in a fiber bundle extending ventrally from Lissauer's tract in lamina I along the lateral edge of the dorsal horn to the sacral parasympathetic nucleus in the L6-S1

spinal segments (lateral collateral pathway of Lissauer). After chronic cystitis, PACAP immunoreactivity in cells in the L1, L2, L6, and S1 DRG increased significantly (P ltoreq 0.0001), and the percentage of bladder afferent cells expressing PACAP immunoreactivity also increased significantly (P ltoreq 0.0001; 70-85%). No changes were observed in the L3-L5 DRG. These studies suggest that the neuropeptide, PACAP, may play a role in urinary bladder afferent pathways after visceral (urinary bladder) inflammation. Changes in PACAP expression after cystitis may play a role in altered visceral sensation (allodynia) and/or urinary bladder hyperreflexia in the clinical syndrome, interstitial cystitis.

L5 ANSWER 23 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2001:96859 BIOSIS
DN PREV200100096859
TI Alterations in urothelium and bladder afferents in feline interstitial cystitis.
AU Buffington, C. A. [Reprint author]; Kiss, S.; Kanai, A. J.; Dineley, K.; Roppolo, J. R.; Reynolds, I. J.; de Groat, W. C.; Birder, L. A.
CS Ohio State University, Columbus, OH, USA
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-349.2. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 21 Feb 2001
Last Updated on STN: 15 Feb 2002
AB The properties of bladder afferent neurons and urothelial cells were examined in normal cats and in cats diagnosed with FIC, a chronic painful disorder of the urinary bladder (UB). The UB, sacral dorsal root ganglia (DRG) and spinal cord (SC), were removed from anesthetized adult cats of either sex before or after perfusion fixation. Small numbers of c-jun-immunoreactive bladder DRG cells were detected in normal cats (<4

cells/section), but the numbers increased (200%) in cats with FIC. UB-DRG cells, labeled by axonal tracers were larger (25%) in FIC cats. The density of substance P and CGRP containing afferent nerves in the UB and spinal dorsal horn was greater in FIC cats. Basal nitric oxide (NO) release, measured with a microsensor in bladder strips, was detected in FIC cats but not in normal cats, whereas NO release evoked by capsaicin was decreased (60%) in normal cats. The UB of FIC cats displayed regions of denuded uroepithelium as evidenced by changes in cytokeratin staining. In cultured uroepithelial cells intracellular calcium measurements using Fura-2 and fluorescent microscopic techniques revealed that sensitivity to purinergic agents was altered in FIC cats. Activation of P2Y receptors (2-methylthio ATP) increased calcium in normal cats, whereas activation of P2X (alpha,beta methylene ATP) or P2Y receptors was effective in FIC cats. These studies raise the possibility that changes in properties of afferent nerves and/or the urothelium may contribute to the painful symptoms in FIC.

L5 ANSWER 24 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1999:160213 BIOSIS
DN PREV199900160213
TI Autonomous neuropathy in interstitial cystitis.
AU Peeker, Ralph [Reprint author]; Aldenborg, Frank [Reprint author]; Li, Jia-Yi [Reprint author]; Fall, Magnus [Reprint author]; Dahlstrom, Annica [Reprint author]; Johansson, Sonny L.
CS Gothenburg, Sweden
SO Journal of Urology, (April, 1999) Vol. 161, No. 4 SUPPL., pp. 28. print.
Meeting Info.: 94th Annual Meeting of the American Urological Association, Inc. Dallas, Texas, USA. May 1-6, 1999. American Urological Association.
CODEN: JOURAA. ISSN: 0022-5347.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English
ED Entered STN: 16 Apr 1999
Last Updated on STN: 16 Apr 1999

L5 ANSWER 25 OF 27 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights
reserved on STN
AN 1997291246 EMBASE
TI Neurophysiology of micturition and continence in women.
AU Chai, T.C.; Steers, W.D., Prof. (correspondence)
CS University of Virginia Health Sciences Center, Department of Urology,
Charlottesville, VA, United States.
AU Steers, W.D., Prof. (correspondence)
CS University of Virginia Health Sciences Center, Department of Urology, Box
422, Charlottesville, VA 22908, United States.
AU Steers, W.D., Prof. (correspondence)
CS Univ. Virginia Health Sciences Ctr., Department of Urology, Box 422,
Charlottesville, VA 22908, United States.
SO International Urogynecology Journal and Pelvic Floor Dysfunction, (1997)
Vol. 8, No. 2, pp. 85-97.
Refs: 150
ISSN: 0937-3462 CODEN: IUFDFV
CY United Kingdom
DT Journal; General Review; (Review)
FS 010 Obstetrics and Gynecology
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
LA English
SL English
ED Entered STN: 9 Oct 1997
Last Updated on STN: 9 Oct 1997
AB Micturition and continence involve the coordination of complex neural
events between the central and peripheral nervous systems. An
understanding of these events provides a foundation for the treatment of
voiding disorders in women such as stress urinary incontinence, urge
incontinence and interstitial cystitis. The purpose
of this paper is to comprehensively review the neuroanatomy,
neurophysiology and neuropharmacology of micturition and continence.
However, a brief section discussing clinical correlations will follow each
of these topics to help integrate the basic science with clinical
obervations.

L5 ANSWER 26 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN DUPLICATE 10
AN 1992:218126 BIOSIS
DN PREV199293118351; BA93:118351
TI INTERSTITIAL CYSTITIS INCREASED SYMPATHETIC
INNERVATION AND RELATED NEUROPEPTIDE SYNTHESIS.
AU HOHENFELLNER M [Reprint author]; NUNES L; SCHMIDT R A; LAMPEL A; THUEROFF
J W; TANAGHO E A
CS DEP UROL, KLINIKUM BARMEN, HEUSNERSTR 40, 5600 WUPPERTAL, WEST GERMANY
SO Journal of Urology, (1992) Vol. 147, No. 3 PART 1, pp. 587-591.
CODEN: JOURAA. ISSN: 0022-5347.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 4 May 1992
Last Updated on STN: 5 May 1992
AB To investigate the possibility of a neural deterioration of the bladder
wall in interstitial cystitis, bladder tissue from 10
patients with interstitial cystitis was compared with
that from 10 control subjects by means of immunohistochemistry. An
enhanced innervation of the bladder in the submucosa and detrusor muscle
was found to represent an increase of sympathetic but not cholinergic
neurons. In interstitial cystitis the number of
neurons positive for vasoactive intestinal polypeptide and neuropeptide Y
was higher and carried a larger number of axonal varicosities, whereas the
number of neurons positive for substance P and calcitonin-
gene-related peptide was not significantly
different in both groups. We conclude that interstitial
cystitis is associated with increased sympathetic outflow into the
bladder and altered metabolism of vasoactive intestinal polypeptide and
neuropeptide Y. Since similar changes have been observed in other
inflammatory diseases of a presumably autoimmune nature, such as
rheumatoid arthritis, Crohn's disease and colitis ulcerosa, the
pathophysiology of interstitial cystitis may share
common pathways with the latter. Experience in these diseases may
facilitate a better understanding of the pathophysiology of
interstitial cystitis and suggest new therapeutic
concepts.

L5 ANSWER 27 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN
AN 1992:298493 BIOSIS
DN PREV199243010843; BR43:10843
TI IMMUNOHISTOCHEMICAL EXAMINATION OF NEUROPEPTIDES AND M-2 MUSCARINIC
RECEPTORS IN NORMAL AND INTERSTITIAL CYSTITIS IC BLADDERS.
AU SHICKLEY T J [Reprint author]; LUTHIN G F; RUGGIERI M R
CS PHILADELPHIA, PA, USA
SO Journal of Urology, (1992) Vol. 147, No. 4 SUPPL, pp. 462A.
Meeting Info.: AUA (AMERICAN UROLOGICAL ASSOCIATION) EIGHTY-SEVENTH ANNUAL
MEETING, WASHINGTON, D.C., USA, MAY 10-14, 1992. J UROL.
CODEN: JOURAA. ISSN: 0022-5347.
DT Conference; (Meeting)
FS BR
LA ENGLISH
ED Entered STN: 16 Jun 1992
Last Updated on STN: 16 Jun 1992

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FILE 'CAPLUS' ENTERED AT 17:35:03 ON 25 JAN 2010

FILE 'EMBASE' ENTERED AT 17:35:03 ON 25 JAN 2010


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=> s interstitial cystitis
L6        4413 INTERSTITIAL CYSTITIS

=> d his

     (FILE 'HOME' ENTERED AT 17:14:58 ON 25 JAN 2010)

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:15:16 ON 25 JAN 2010
L1        31928 S CALCITONIN GENE RELATED PEPTIDE OR CGRP
L2         9041 S PITUITARY ADENYLATE CYCLASE ACTIVATING PEPTIDE OR
PACAP
L3        40576 S L1 OR L2
L4           43 S L3 AND INTERSTITIAL CYSTITIS
L5           27 DUP REM L4 (16 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 17:23:30 ON 25 JAN 2010

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:35:03 ON 25 JAN 2010
L6         4413 S INTERSTITIAL CYSTITIS

=> s l6 and diagnos?
L7        1735 L6 AND DIAGNOS?

=> s l7 and review
L8         316 L7 AND REVIEW

=> d bib abs 1-10

L8   ANSWER 1 OF 316  BIOSIS  COPYRIGHT (c) 2010 The Thomson
Corporation  on
     STN
AN   2010:6079  BIOSIS
DN   PREV201000006079
TI   Bladder Outlet Obstruction in Painful Bladder Syndrome/
     Interstitial Cystitis.
AU   Cameron, Anne P. [Reprint Author]; Gajewski, Jerzy B.
CS   1500 Med Ctr Dr,3875 Taubman Ctr, Ann Arbor, MI 48109 USA
     annepell@med.umich.edu
SO   Neurourology and Urodynamics, (2009) Vol. 28, No. 8, pp.
944-948.
     CODEN: NEUREM. ISSN: 0733-2467.
DT   Article
```

LA English
ED Entered STN: 9 Dec 2009
Last Updated on STN: 9 Dec 2009
AB Aims: Obstructive symptoms such as slow stream, dribbling and straining
are often reported by painful bladder syndrome and interstitial
cystitis (PBS/IC) patients. Our hypothesis was that some patients
with PBS/IC have an associated measurable bladder outlet obstruction (BOO)
secondary to dysfunctional voiding and that those patients with more
severe PBS/IC are more likely to have BOO. Methods: This is a
retrospective chart review of female patients diagnosed
with PBS/IC based on the NIDDK research definition. Charts were reviewed
for clinical symptom severity, ulcer or non-ulcer PBS/IC on cystoscopy,
and pressure-flow urodynamics (UDPF). Patients were excluded if they had
a urinary infection at the time of urodynamics or did not meet study entry
requirements. The cut-off values of <= 12 ml/sec and >= 25 cm of water
was used to define BOO. Results: Of the 231 women: 38 had ulcer PBS/IC
and 193 had non-ulcer PBS/IC. MCC was 269 ml in non-ulcer PBS/IC and 200
ml in ulcer PBS/IC (P = 0.006). One hundred eleven women (48%) met
criteria for obstruction. MCC was 298 ml in the non-obstructed group and
214 ml in the obstructed group (P < 0.0001). The maximum flow with
non-ulcer PBS/IC was 11.0 ml/sec and in ulcer PBS/IC 8.9 ml/sec (P = 0.04)
Detrusor pressure at maximum flow was 33.3 cm H2O, in non-ulcer, and 37.4
cm H2O in ulcer PBS/IC (P = 0.01). Conclusions: Forty-eight percent of
our PBS/IC patients have BOO, and increasing severity of PBS/IC is
associated with higher voiding pressure. Neurourol. Urodynam.
28:944-948, 2009. (C) 2009 Wiley-Liss, Inc.

L8 ANSWER 2 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN
AN 2009:298223 BIOSIS
DN PREV200900299326
TI Developmental Influences on Medically Unexplained Symptoms.
AU Buffington, C. A. Tony [Reprint Author]

CS Ohio State Univ, Vet Hosp, 601 Tharp St, Columbus, OH 43210 USA
Buffington.1@osu.edu
SO Psychotherapy and Psychosomatics, (2009) Vol. 78, No. 3, pp. 139-144.
CODEN: PSPSBF. ISSN: 0033-3190.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 6 May 2009
Last Updated on STN: 20 May 2009
AB Background: Medically unexplained (or 'functional') symptoms (MUS) are physical symptoms that prompt the sufferer to seek healthcare but remain unexplained after an appropriate medical evaluation. Examples of MUS also occur in veterinary medicine. For example, domestic cats suffer a syndrome comparable to interstitial cystitis, a chronic pelvic pain syndrome of humans. Method: Review of current evidence suggests the hypothesis that developmental factors may play a role in some cases of MUS. Maternal perception of a threatening environment may be transmitted to the fetus when hormones cross the placenta and affect fetal physiology, effectively 'programming' the fetal stress response system and associated behaviors toward enhanced vigilance. After birth, intense stress responses in the individual may result in similar vulnerability, which may be unmasked by subsequent stressors. Results: Epigenetic modulation of gene expression (EMGEX) appears to play a central role in creation of this 'survival phenotype'. The recent development of techniques to identify the presence of EMGEX provides new tools to investigate these questions, and drugs and other interventions that may reverse EMGEX are also under active investigation. Conclusion: Viewing MUS from the perspective of underlying developmental influences involving EMGEX that affect function of a variety of organs based on familial (genetic and environmental) predispositions rather than from the traditional viewpoint of isolated organoriginating diseases has at least

two important implications: it provides a parsimonious explanation for findings heretofore difficult to reconcile, and it opens whole new areas of investigation into causes and treatments for this class of disorders.
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L8 ANSWER 3 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2009:185242 BIOSIS
DN PREV200900185242
TI Breaking the Cycle of Pain in Interstitial Cystitis /Painful Bladder Syndrome Toward Standardization of Early Diagnosis and Treatment.
AU Forrest, John B. [Reprint Author]; Mishell, Daniel R. Jr.
CS 10901 E 48th St S, Tulsa, OK 74146 USA
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SO Journal of Reproductive Medicine, (JAN 2009) Vol. 54, No. 1, pp. 3-14.
CODEN: JRPMAP. ISSN: 0024-7758.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 11 Mar 2009
Last Updated on STN: 11 Mar 2009
AB Chronic pelvic pain (CPP) affects about 15% of female adults in the United States. The source of this pain in many women is the bladder, specifically interstitial cystitis/painful bladder syndrome (IC/PBS). Despite the frequent occurrence of IC/PBS as a cause of CPP, there currently are no universally accepted guidelines for diagnosis and treatment of this disorder, and, consequently, many patients do not receive appropriate treatment in a timely manner. In an effort to develop a rational way to diagnose and treat patients With CPP, a panel of leaders in urology, gynecology, urogynecology and general women's health met to review recent literature, reach consensus and formulate 2 algorithms, one for diagnosing and the other for managing IC/PBS. This article reflects the results of that meeting. (J Reprod Med 2009;54:3-14)

L8 ANSWER 4 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2009:162050 BIOSIS
DN PREV200900162050

TI The Spectrum of Eosinophilic Cystitis in Males Case Series and Literature Review.
AU Popescu, Oana-Eugenia; Landas, Steve K. [Reprint Author]; Haas, Gabriel P.
CS State Univ New York Upstate Med Univ, Dept Pathol and Urol, 750 E Adams St, Syracuse, NY 13210 USA
landas@upstate.edu
SO Archives of Pathology & Laboratory Medicine, (FEB 2009) Vol. 133, No. 2, pp. 289-294.
CODEN: APLMAS. ISSN: 0003-9985.
DT Article
LA English
ED Entered STN: 4 Mar 2009
Last Updated on STN: 4 Mar 2009
AB Context.-Eosinophilic cystitis (EC) is an inflammatory condition of the bladder that has been linked to food allergens, infectious agents, drugs, and other genitourinary conditions. Like interstitial cystitis, EC has a strong female predominance. It is characterized by an intense eosinophilic infiltrate in the acute phase and fibrosis in the chronic phase.Objectives.-To document and focus on specific features of EC in males and highlight the relationship between clinical and histopathologic findings.Design.-The bladder biopsies of male patients were reviewed. Eight cases of EC were selected.Results.-Several known associations were noted as well as unreported features and associations such as Charcot-Leyden crystals, celiac disease, lupus anticoagulant, and additional viral and bacterial agents.Conclusions.-Eosinophilic cystitis represents a response to a variety of agents and may often be overlooked. The temporally biphasic morphologic features are the hallmark of this condition. Because clinical and imaging studies are not specific, a high index of clinical suspicion is often crucial to the correct diagnosis and proper management of EC.

L8 ANSWER 5 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2008:538440 BIOSIS

DN PREV200800538439
TI Disorders of adhesions or adhesion-related disorder: Monolithic entities
or part of something bigger - CAPPS?.
AU Wiseman, David M. [Reprint Author]
CS Int Adhes Society, PMB 238,6757 Arapaho,Suite 711-238, Dallas, TX 75248
USA
david.wiseman@adhesions.org
SO Seminars in Reproductive Medicine, (JUL 2008) Vol. 26, No. 4, pp. 356-368.
ISSN: 1526-8004.
DT Article
LA English
ED Entered STN: 1 Oct 2008
Last Updated on STN: 1 Oct 2008
AB The purpose of this article is to review progress in the field of abdominopelvic adhesions and the validity of its two underlying assumptions: (1) The formation of adhesions results in infertility, bowel obstruction, or other complications. Reducing or avoiding adhesions will curb these sequelae. (2) "Adhesions" is a monolithic entity to be tackled without regard to any other condition.Evidence is discussed to validate the first assumption. We reviewed progress in the field by examining hospital data. We found a growing trend in the number and cost of discharges for just two adhesion-related diagnoses, and the low usage of adhesion barriers appears in at most 5% of appropriate procedures. Data from an Internet-based survey suggested that the problem may, be partly due to ignorance among patients and physicians about adhesions and their prevention.Two other surveys of patients visiting the adhesions.org Web site defined more fully adhesion-related disorder (ARD).
The first survey (N= 466) described a patient with chronic pain, gastrointestinal disturbances, an average of nine bowel obstructions, and an inability to work or maintain family or social relationships. The second survey (687 U.S. women) found a high (co-) prevalence of abdominal or pelvic adhesions (85%), chronic abdominal or pelvic pain (69%), irritable bowel syndrome (55%), recurrent bowel obstruction (44%),

endometriosis (40%), and interstitial cystitis (29%).This pattern Suggests that although "adhesions" may, start out as a monolithic entity, an adhesions patient may develop related conditions (ARD) until they merge into an independent entity where they are practically indistinguishable from patients with multiple symptoms originating from other abdominopelvic conditions such as pelvic or bladder pain. Rather than use terms that constrain the required multidisciplinary, biopsychosocial approach to these patients by the paradigms of the specialty related to the patient's initial symptom set, the term complex abdominopelvic and pain syndrome (CAPPS) is proposed. It is essential to understand not only the pathogenesis of the "initiating" conditions but also how they progress to CAPPS. In our ARD sample, not only was the frequency of women with hysterectomies (56%) higher than expected (21 to 33%), but also the rates of the "initiating" conditions was 40 to 400% higher in patients with hysterectomies than in those without. This may represent increased surgical trauma or the loss of protection against oxidative stress. Related was the higher frequency of ARD patients reporting hemochromatosis (HC; 5%) than expected (similar to 0.5%) and the higher rates (20 to 700%) of initiating conditions in patients with HC than in those without HC. Together with findings related to the toxicity of Intergel, these findings raise the possibility, that heterozygotes for genes regulating oxidative stress are at greater risk of developing surgical complications as well as more severe and progressive conditions such as CAPPS.

L8 ANSWER 6 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2008:395348 BIOSIS
DN PREV200800395347
TI Urinary tract infection and inflammation at onset of interstitial cystitis/painful bladder syndrome.

AU Warren, John W. [Reprint Author]; Brown, Vivian; Jacobs, Stephen; Horne,
Linda; Langenberg, Patricia; Greenberg, Patty
CS Univ Maryland, Sch Med, Dept Med, 10 S Pine St,Room 9-00 MSTF, Baltimore,
MD 21201 USA
Studyic@medicine.umaryland.edu
SO Urology, (JUN 2008) Vol. 71, No. 6, pp. 1085-1090.
ISSN: 0090-4295.
DT Article
LA English
ED Entered STN: 16 Jul 2008
Last Updated on STN: 16 Jul 2008
AB OBJECTIVES Interstitial cystitis/painful bladder syndrome (IC/PBS) is a chronic disease primarily in women that is of low incidence and, unknown etiology and manifests as bladder pain and urinary symptoms. Acute urinary tract infection (UTI) is of high incidence in women, presents as dysuria and urinary symptoms, and is caused by uropathogenic bacteria. We hypothesized that UTI is present at the onset of IC/PBS in some women.METHODS For a case-control study seeking risk factors for IC/PBS, women with IC/PBS symptoms of 12 months or less were recruited and evaluated by interview and medical record review. The date of symptom onset was identified by a six-step process. Three evidence-based methods using culture, urinalysis, and symptoms were used separately and in combination to diagnose UTI at IC/PBS onset.RESULTS Of 1177 screened women, 314 with recent-onset IC/PBS, including numerous confirming characteristics, were enrolled in the study; 98% of the requested medical records were obtained and reviewed. Evidence of a UTI at the onset of IC/PBS was found in 18% to 36% of women. Common UTI features not used in its diagnosis (short interval to medical care, hematuria, antibiotic treatment, and improvement after antibiotics) were significantly more common in those with onset UTI than in those without.CONCLUSIONS These retrospective data suggest that a proportion, probably a minority, of women at IC/PBS onset had evidence of UTI or inflammation. Our results indicate that UTI is present at the

onset of IC/PBS in some women and might reveal clues to IC/PBS pathogenesis.

L8 ANSWER 7 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2008:304655 BIOSIS
DN PREV200800306707
TI Characterization of a clinical cohort of 87 women with interstitial cystitis/painful bladder syndrome.
AU Peters, Kenneth M.; Carrico, Donna J. [Reprint Author]; Diokno, Ananias C.
CS William Beaumont Hosp, Dept Urol, Ministrelli Program Urol Res and Educ, 3535 W 13 Mile Rd,Suite 438, Royal Oak, MI 48073 USA dcarrico@beaumont.edu
SO Urology, (APR 2008) Vol. 71, No. 4, pp. 634-640. ISSN: 0090-4295.
DT Article
LA English
ED Entered STN: 12 May 2008 Last Updated on STN: 12 May 2008
AB OBJECTIVE To provide a characterization of a cohort of women with interstitial cystitis/painful bladder syndrome (IC/PBS) by describing their historical and clinical characteristics. This was reported with the National Institutes of Health chronic prostatitis cohort, but a literature review did not reveal a similar study for women with IC/PBS.METHODS A total of 87 women with IC/PBS were referred to the Beaumont Women's Initiative for Pelvic Pain and Sexual Health program. A certified nurse practitioner took a comprehensive history and per-formed a pelvic exam for each. Data were analyzed using descriptive statistics to describe this cohort.RESULTS Most women experienced constant pain for 5 or more years (mean Visual Analog Scale = 5 out of 10). A total of 94.2% had levator pain. More than 50% had vulvar pain with exam. More than half reported a history of abuse, often in more than one life stage. A total of 28% had cesarean births and 76% had a history of miscarriage, stillbirth, or abortion. Women averaged 4 lifetime pelvic surgeries, and 48% had hysterectomies, two-thirds of which were done before IC/PBS diagnosis. Premenstrual women reported pain throughout the menstrual cycle. As many as 12% had chlamydia

previously, which was higher than the national average. Common comorbidities were pelvic pain (93%), allergies (86%), and sexual dysfunction (72%).CONCLUSIONS This population of women with unrelieved chronic pain, frequency, and urgency is in desperate need of care. Researchers should continue to search for the etiology, prevention, and treatment interventions that are effective in dealing with IC/PBS. It may be most therapeutic to develop a multimodal plan of care that includes physical therapy, oral and intravesical therapies, neuromodulation, and cognitive-behavioral therapies.

L8 ANSWER 8 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2008:139703 BIOSIS
DN PREV200800138354
TI Reprogramming requirements after sacral nerve stimulator implantation: Correlation with preoperative indication.
AU Maxwell, Kelly M.; Clemens, J. Quentin; Mazzenga, Laura; Kielb, Stephanie J. [Reprint Author]
CS Northwestern Univ, Feinberg Sch Med, Dept Urol, 303 E Chicago Ave,Tarry 16-703, Chicago, IL 60611 USA skielb@nmff.org
SO Journal of Urology, (FEB 2008) Vol. 179, No. 2, pp. 549-551. CODEN: JOURAA. ISSN: 0022-5347.
DT Article
LA English
ED Entered STN: 20 Feb 2008 Last Updated on STN: 20 Feb 2008
AB Purpose: Recent publications support sacral nerve stimulator implantation in patients with interstitial cystitis. To our knowledge the reprogramming requirements for all patients following stimulator implantation has not been described and it is unknown whether the number of sessions required vary by pre-implantation diagnosis . We determined overall reprogramming requirements following nerve stimulator implantation and whether requirements vary based on preoperative indication.Materials and Methods: After obtaining institutional review board approval we retrospectively reviewed the records of all patients who underwent sacral nerve stimulator

implantation at our institution between June 2002 and October 2004. The preoperative indication and number of reprogramming sessions during the initial test period (stage 1) and following permanent implantation (stage 2) were compared.Results: The 17 patients proceeding to stage 2 with a minimum 12-month followup during the study period were included. Mean age was 43 years (range 26 to 78) and all patients except 1 were female. Patients were separated by diagnosis for evaluation purposes, including urgency/frequency/incontinence in 8, urinary retention in 2 and interstitial cystitis in 7. The average number of reprogramming sessions during stage 1 was 0.9, 3.5 and 2.3 for urgency/frequency/incontinence, urinary retention and interstitial cystitis, respectively. The average number of reprogramming sessions after stage 2 was 2.8, 3.0 and 6.9 at 12-month followup for urgency/frequency/incontinence, urinary retention and interstitial cystitis, respectively. No patient had the stimulator removed for reprogramming failure.Conclusions: Patients in urinary retention appear to require more frequent reprogramming during stage 1, while patients with interstitial cystitis require more sessions after stage 2 implantation.

L8 ANSWER 9 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2008:85285 BIOSIS
DN PREV200800078702
TI Pharmacologic management of painful bladder syndrome/interstitial cystitis: A systematic review (vol 167, pg 1922, 2007).
AU Dimitrakov, J.; Kroenke, K.; Steers, W. D.; Berde, C.; Zurakowski, D.; Freeman, M. R.; Jackson, J. L.
SO Archives of Internal Medicine, (DEC 10 2007) Vol. 167, No. 22, pp. 2452. CODEN: AIMDAP. ISSN: 0003-9926.
DT Article Errata
LA English
ED Entered STN: 23 Jan 2008 Last Updated on STN: 23 Jan 2008

L8 ANSWER 10 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN
AN 2008:35914 BIOSIS
DN PREV200800035088
TI Pharmacologic management of painful bladder syndrome/interstitial cystitis - A systematic review.
AU Dimitrakov, Jordan [Reprint Author]; Kroenke, Kurt; Steers, William D.;
Berde, Charles; Zurakowski, David; Freeman, Michael R.; Jackson, Jeffrey
L.
CS Harvard Univ, Sch Med, Childrens Hosp Boston, Harvard Urol Dis Res Ctr,
Enders Res Bldg,Room 1061,300 Longwood Ave, Boston, MA 02115 USA
Jordan.Dimitrakov@childrens.harvard.edu
SO Archives of Internal Medicine, (OCT 8 2007) Vol. 167, No. 18, pp. 1922-1929.
CODEN: AIMDAP. ISSN: 0003-9926.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 27 Dec 2007
Last Updated on STN: 27 Dec 2007
AB Background: More than 180 different types of therapy have been used in the
treatment and management of painful bladder syndrome/interstitial cystitis (PBS/IC), yet evidence from clinical trials remains inconclusive. This study aimed to evaluate the efficacy of pharmacologic
approaches to PBS/IC, to quantify the effect size from randomized controlled trials, and to begin to inform a clinical consensus of treatment efficacy for PBS/IC.Methods: We identified randomized controlled
trials for the pharmacologic treatment of patients wth PBS/IC diagnosed on the basis of National Institute of Diabetes and Digestive and Kidney Diseases or operational criteria. Study limitations
include considerable patient heterogeneity as well as variability in the
definition of symptoms and in outcome assessment.Results: We included a
total of 1470 adult patients from 21 randomized controlled trials. Only
trials for pentosan polysulfate sodium had sufficient numbers to allow a
pooled analysis of effect. According to a random-effects model, the
pooled estimate of the effect of pentosan polysulfate therapy suggested
benefit, with a relative risk of 1.78 for patient-reported improvement in

symptoms (95% confidence interval, 1.34-2.35). This result was not heterogeneous (P=.47) and was without evidence of publication bias (P =.18). Current evidence also suggests the efficacy of dimethyl sulfoxide and amitryp- tiline therapy. Hydroxyzine, intravesical bacille Calmette-Guerin, and resiniferatoxin therapy failed to demonstrate efficacy, but evidence was inconclusive owing to methodological limitations.Conclusions: Pentosan polysulfate may be modestly beneficial for symptoms of PBS/IC. There is insufficient evidence for other pharmacologic treatments. A consensus on standardized outcome measures is urgently needed.

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.85

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LAST RELOADED: Jan 22, 2010 (20100122/UP).

=> FIL BIOSIS CAPLUS EMBASE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	171.96

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.85

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=> d his

     (FILE 'HOME' ENTERED AT 17:14:58 ON 25 JAN 2010)

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:15:16 ON 25 JAN 2010
L1        31928 S CALCITONIN GENE RELATED PEPTIDE OR CGRP
L2         9041 S PITUITARY ADENYLATE CYCLASE ACTIVATING PEPTIDE OR
PACAP
L3        40576 S L1 OR L2
L4           43 S L3 AND INTERSTITIAL CYSTITIS
L5           27 DUP REM L4 (16 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 17:23:30 ON 25 JAN 2010

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:35:03 ON 25 JAN 2010
L6         4413 S INTERSTITIAL CYSTITIS
L7         1735 S L6 AND DIAGNOS?
L8          316 S L7 AND REVIEW

     FILE 'STNGUIDE' ENTERED AT 17:39:36 ON 25 JAN 2010

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:41:39 ON 25 JAN 2010

=> s l3 and pelvic pain
L9          19 L3 AND PELVIC PAIN

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10          12 DUP REM L9 (7 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 12 ANSWERS - CONTINUE? Y/(N):y

L10  ANSWER 1 OF 12  EMBASE  COPYRIGHT (c) 2010 Elsevier B.V. All
rights
     reserved on STN
AN   2009546163  EMBASE
TI   Visceral hyperalgesia in chronic pelvic pain.
AU   Aslam, N.; Harrison, G.; Khan, K.
CS   Academic Department of Obstetrics and Gynaecology, Birmingham
University
     Hospital, Birmingham, United Kingdom.
AU   Patwardhan, S. (correspondence)
CS   Walsgrave University Hospital, Clifford Bridge Road, Coventry
CV2 2DX,
     United Kingdom. drsanjaypatwardhan@gmail.com
SO   BJOG: An International Journal of Obstetrics and Gynaecology,
(November
```

2009) Vol. 116, No. 12, pp. 1551-1555.
Refs: 25
ISSN: 1470-0328; E-ISSN: 1471-0528 CODEN: BIOGFQ
PB Blackwell Publishing Ltd, 9600 Garsington Road, Oxford, OX4 2XG, United
Kingdom.
CY United Kingdom
DT Journal; Note
FS 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
010 Obstetrics and Gynecology
028 Urology and Nephrology
037 Drug Literature Index
LA English
ED Entered STN: 18 Nov 2009
Last Updated on STN: 18 Nov 2009

L10 ANSWER 2 OF 12 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights
reserved on STN
AN 2009489895 EMBASE
TI Endometriosis-associated nerve fibers and pain.
AU Medina, Melissa G.
CS University of Michigan, Ann Arbor, MI, United States.
AU Lebovic, Dan I.
CS Department of Obstetrics and Gynecology, Division of Reproductive
Endocrinology and Infertility, University of Wisconsin, Madison, WI,
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AU Lebovic, Dan I.
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United States. Lebovic@wisc.edu
AU Lebovic, D. I. (correspondence)
CS Department of Obstetrics and Gynecology, University of Wisconsin, H4/628
Clinical Science Center, 600 Highland Avenue, Madison, WI 53792-3236,
United States. Lebovic@wisc.edu
SO Acta Obstetricia et Gynecologica Scandinavica, (2009) Vol. 88, No. 9, pp.
968-975.
Refs: 29
ISSN: 0001-6349; E-ISSN: 1600-0412 CODEN: AOGSAE
PB Informa Healthcare, Telephone House, 69 - 77 Paul Street, EC2A 4LQ, United
Kingdom.
PUI 913657353
CY United Kingdom
DT Journal; General Review; (Review)

FS 005 General Pathology and Pathological Anatomy
010 Obstetrics and Gynecology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 11 Nov 2009
Last Updated on STN: 11 Nov 2009
AB The assessment and diagnosis of endometriosis remain elusive targets.
Patient and medical-related factors add to delays in the detection and
treatment. Recently, investigators have revealed specific nerve fibers
present in endometriotic tissue, with existing parallels between density
and pain severity. The aim of this review is to compile a comprehensive
review of existing literature on endometriosis-related nerve fiber
detection, and the effects of medical therapy on these neural fibers. We
performed a systematic literature-based review using Medline and PubMed of
nerve fibers detected in eutopic endometrium, endometriotic lesions, and
the peritoneum. Various arrangements of significant medical terms and
phrases consisting of endometriosis, pelvic pain,
nerve fiber detection/density in endometriosis, and diagnoses methodology,
including treatment and detection were applied in the search. Subsequent
references used were cross-matched with existing sources to compile all
additional similar reports. Similar nerve fibers were detected within
lesions, endometrium, and myometrium, though at varying degrees of
density. Hormonal therapy is widely used to treat endometriosis and was
shown to be related to a reduction in fiber density. A direct result of
specific nerve fiber detection within eutopic endometrial layers points to
the use of a minimally invasive endometrial biopsy technique in reducing
delay in diagnosis and subsequent possible preservation of fertility.

L10 ANSWER 3 OF 12 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 1

AN 2009:263278 BIOSIS
DN PREV200900263278
TI Rich innervation of deep infiltrating endometriosis.
AU Wang, Guoyun; Tokushige, Natsuko [Reprint Author]; Markham, Robert;
Fraser, Ian S.
CS Univ Sydney, Queen Elizabeth II Res Inst Mothers and Infants, Dept Obstet
and Gynecol, Sydney, NSW 2006, Australia
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SO Human Reproduction (Oxford), (APR 2009) Vol. 24, No. 4, pp. 827-834.
CODEN: HUREEE. ISSN: 0268-1161.
DT Article
LA English
ED Entered STN: 16 Apr 2009
Last Updated on STN: 16 Apr 2009
AB Deep infiltrating endometriosis (DIE) is a specific type of endometriosis,
which can be associated with more severe pelvic pain
than other forms of endometriotic lesions. However, the mechanisms by
which pain is generated are not well understood.DIE (n = 31) and
peritoneal endometriotic (n = 40) lesions were sectioned and stained
immunohistochemically with antibodies against protein gene product 9.5,
neurofilament, nerve growth factor (NGF), NGF receptors tyrosine kinase
receptor-A (Trk-A) and p75, substance P, calcitonin gene
-related peptide, vesicular acetylcholine transporter,
neuropeptide Y, vasoactive intestinal peptide and tyrosine hydroxylase to
demonstrate myelinated, unmyelinated, sensory and autonomic nerve
fibres.There were significantly more nerve fibres in DIE (67.6 +/-
65.1/mm(2)) than in peritoneal endometriotic lesions (16.3 +/- 10.0/mm(2))
(P < 0.01). DIE was innervated abundantly by sensory A delta, sensory C,
cholinergic and adrenergic nerve fibres; NGF, Trk-A and p75 were strongly
expressed in endometriotic glands and stroma of DIE.The rich innervation
of DIE may help to explain why patients with this type of lesion have
severe pelvic pain.

L10 ANSWER 4 OF 12 EMBASE
AN 2008361822 EMBASE

TI Evidence for the use of botulinum toxin in the chronic pain setting - A review of the literature.
AU Jeynes, Louise C.
CS The Boyle Department of Anesthesia, St. Bartholomew's Hospital, London, United Kingdom.
AU Gauci, Charles A., Dr. (correspondence)
CS Whipps Cross University Hospital, London, United Kingdom. charles.gauci@btinternet.com
AU Gauci, Charles A., Dr. (correspondence)
CS Queen's Hospital, Essex, United Kingdom. charles.gauci@btinternet.com
SO Pain Practice, (July/August 2008) Vol. 8, No. 4, pp. 269-276.
Refs: 100
ISSN: 1530-7085; E-ISSN: 1533-2500 CODEN: PPARCJ
PB Blackwell Publishing Inc., 350 Main Street, Malden, MA 02148, United States.
CY United States
DT Journal; (Short Survey)
FS 008 Neurology and Neurosurgery
019 Rehabilitation and Physical Medicine
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
ED Entered STN: 7 Aug 2008
Last Updated on STN: 7 Aug 2008
AB A significant proportion of chronic pain is of musculoskeletal origin.
Botulinum toxin (BTX) has been successfully used in the treatment of spasmodic torticollis, limb dystonia, and spasticity. Investigators have, thus, become interested in its potential use in treating many chronic pain conditions. Practitioners have used BTX, outside the product license, in the treatment of refractory myofascial pain syndrome and neck and low back pain (LBP). This article reviews the current evidence relating to chronic pain practice. There is evidence supporting the use of both BTX type A and type B in the treatment of cervical dystonias. The weight of evidence is in favor of BTX type A as a treatment in: pelvic pain, plantar fasciitis, temporomandibular joint dysfunction associated facial

pain, chronic LBP, carpal tunnel syndrome, joint pain, and in complex regional pain syndrome and selected neuropathic pain syndromes. The weight of evidence is also in favor of BTX type A and type B in piriformis syndrome. There is conflicting evidence relating to the use of BTX in the treatment whiplash, myofascial pain, and myogenous jaw pain. It does appear that BTX is useful in selected patients, and its duration of action may exceed that of conventional treatments. This seems a promising treatment that must be further evaluated. .COPYRGT. 2008 World Institute of Pain.

L10 ANSWER 5 OF 12 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2007507114 EMBASE
TI Neuroendocrine-immune disequilibrium and endometriosis: An interdisciplinary approach.
AU Tariverdian, Nadja; Blois, Sandra M.; Arck, Petra C. (correspondence)
CS Center of Internal Medicine and Dermatology, Division of PsychoNeuroImmunology, University Medicine Berlin, Berlin, Germany.
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AU Theoharides, Theoharis C.
CS Department of Pharmacology and Experimental Therapeutics, Tufts University School of Medicine, Boston, MA, United States.
AU Siedentopf, Friederike
CS Department of Obstetrics and Gynaecology, DRK-Kliniken Westend, Berlin, Germany.
AU Gutierrez, Gabriela
CS Institute of Humoral Immunity Studies-IDEHU (CONICET-UBA), School of Pharmacy and Biochemistry, University of Buenos Aires, Buenos Aires, Argentina.
AU Jeschke, Udo
CS Department of Obstetrics and Gynaecology, Ludwig-Maximilians-University Munich, Munich, Germany.
AU Rabinovich, Gabriel A.
CS Institute of Biology and Experimental Medicine, IBYME-CONICET, Buenos Aires, Argentina.

AU Rabinovich, Gabriel A.
CS Faculty of Exact and Natural Sciences, University of Buenos Aires, Buenos
Aires, Argentina.
AU Arck, Petra C. (correspondence)
CS Biomedizinisches Forschungszentrum, Charite, Campus Virchow,
Augustenburger Platz 1, Berlin 13353, Germany.
petra.arck@charite.de
SO Seminars in Immunopathology, (Jun 2007) Vol. 29, No. 2, pp. 193-210.
Refs: 196
ISSN: 1863-2297; E-ISSN: 1863-2300
CY Germany
DT Journal; General Review; (Review)
FS 010 Obstetrics and Gynecology
026 Immunology, Serology and Transplantation
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 2 Nov 2007
Last Updated on STN: 2 Nov 2007
AB Endometriosis, a chronic disease characterized by endometrial tissue
located outside the uterine cavity, affects one fourth of young women and
is associated with chronic pelvic pain and
infertility. However, an in-depth understanding of the pathophysiology
and effective treatment strategies of endometriosis is still largely
elusive. Inadequate immune and neuroendocrine responses are significantly
involved in the pathophysiology of endometriosis, and key findings are
summarized in the present review. We discuss here the role of different
immune mechanisms particularly adhesion molecules, protein-glycan
interactions, and pro-angiogenic mediators in the development and
progression of the disease. Finally, we introduce the concept of
endometrial dissemination as result of a neuroendocrine-immune
disequilibrium in response to high levels of perceived stress caused by
cardinal clinical symptoms of endometriosis. .COPYRGT. 2007
Springer-Verlag.

L10 ANSWER 6 OF 12 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 2
AN 2007:39911 BIOSIS
DN PREV200700041566
TI Nerve fibres in peritoneal endometriosis.
AU Tokushige, Natsuko [Reprint Author]; Markham, Robert; Russell, Peter;

Fraser, Ian S.
CS Univ Sydney, Dept Obstet and Gynaecol, Queen Elizabeth Res Inst Mothers
and Infants 2, Sydney, NSW 2006, Australia
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SO Human Reproduction (Oxford), (NOV 2006) Vol. 21, No. 11, pp. 3001-3007.
CODEN: HUREEE. ISSN: 0268-1161.
DT Article
LA English
ED Entered STN: 3 Jan 2007
Last Updated on STN: 3 Jan 2007
AB BACKGROUND: Endometriosis is a gynaecological disease that can be associated with severe pelvic pain; however, the mechanisms by which pain is generated remain unknown. METHODS: Peritoneal endometriotic lesions and normal peritoneum were prepared from women with and without endometriosis (n = 40 and 36, respectively). Specimens were also prepared from endosalpingiosis lesions (n = 9). These sections were stained immunohistochemically with antibodies against protein gene product 9.5, neurofilament (NF), nerve growth factor (NGF), NGF receptor p75 (NGFRp75), substance P (SP), calcitonin gene-related peptide (CGRP), acetylcholine (ACh) and tyrosine hydroxylase (TH) to demonstrate myelinated, unmyelinated, sensory, cholinergic and adrenergic nerve fibres. RESULTS: There were significantly more nerve fibres identified in peritoneal endometriotic lesions than in normal peritoneum ($P < 0.001$) or endosalpingiosis lesions ($P < 0.001$). These nerve fibres were SP, CGRP, ACh or TH immunoreactive. Many of these markers were co-localized. There was an intense NGF immunoreactivity near endometriotic glands, and NGFRp75 immunoreactive nerve fibres were present near endometriotic glands and blood vessels in the peritoneal endometriotic lesions. CONCLUSIONS: Peritoneal endometriotic lesions were innervated by sensory A delta, sensory C, cholinergic and adrenergic nerve fibres. These nerve fibres may play an important role in the mechanisms of pain generation in this condition.

L10 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:405354 CAPLUS
DN 142:461621
TI Detection of neuropeptides associated with pelvic pain disorders and uses for diagnosis and treatment
IN Wood, Ronald W.; Reeder, Jay; Schwarz, Edward M.; Messing, Edward M.; Schoen, Susan R.; Vizzard, Margaret A.; Dickerson, Ian
PA University of Rochester, USA
SO PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005041757	A2	20050512	WO 2004-US36015	20041029
WO 2005041757	A3	20060601		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 20080070239	A1	20080320	US 2007-577395	20070427
PRAI US 2003-515408P	P	20031029		
WO 2004-US36015	W	20041029		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The present invention relates generally to the diagnosis and treatment of pelvic pain disorders, including bladder disorders that are characterized by increased expression of the neuropeptides

CGRP and/or PACAP. One aspect of the present invention is directed to a method of diagnosing pelvic pain disorders. This method involves measuring a level of one or both of the neuropeptides calcitonin gene-related peptide (CGRP) or pituitary adenylate cyclase activating peptide (PACAP) in a patient sample and then determining whether the CGRP or PACAP level in the patient sample is elevated in relation to a level of CGRP or PACAP in a normal asymptomatic population. A second aspect of the present invention is directed to a method of determining predisposition of an individual to conditions associated with or development of pelvic pain syndromes. A third aspect of the present invention is directed to a method of treating a pelvic pain disorder in a patient. This method involves providing a CGRP or PACAP antagonist and administering the CGRP or PACAP antagonist to the patient in an amount effective to treat the pelvic pain disorder. A fourth aspect of the present invention is directed to a method of characterizing response to treatment for a pelvic pain disorder. A fifth aspect of the present invention relates to a transgenic nonhuman mammal that includes a first DNA construct that is expressed in bladder sensory neurons, the first DNA construct having a promoter operatively coupled to a DNA mol. encoding a neuropeptide (either PACAP or CGRP). The transgenic nonhuman mammals are characterized by overexpression (i.e., relative to nontransgenic mammals) of the neuropeptide. These transgenic animals are useful for the study of pelvic pain disorders and assessing the efficacy of potential therapeutic agents in the treatment thereof.
OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L10 ANSWER 8 OF 12 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 3
AN 2006:27247 BIOSIS
DN PREV200600024144
TI Possible mechanism of referred pain in the perineum and pelvis associated with the prostate in rats.
AU Chen, Yong; Song, Bo [Reprint Author]; Jin, Xi-Yu; Xiong, En-Qing; Zhang, Jian-Hua
CS Third Mil Med Univ, SW Hosp, Dept Urol, Chongqing 40008, Peoples R China

SO Journal of Urology, (DEC 2005) Vol. 174, No. 6, pp. 2405-2408.
CODEN: JOURAA. ISSN: 0022-5347.
DT Article
LA English
ED Entered STN: 21 Dec 2005
Last Updated on STN: 21 Dec 2005
AB Purpose: Since persistent pain in the perineum and pelvic floor associated
with chronic prostatitis/chronic pelvic pain syndrome
has been hypothesized to be referred pain, it might also be explained by
neural mechanisms.Materials and Methods: Dual retrograde fluorescent
labeling and immunohistochemistry were identified as methods with which to
investigate the neurogenic aspect of this status. The dual distribution
of dorsal root ganglia (DRG) cells was determined after double retrograde
fluorescent staining of the prostate and pelvic floor, and the prostate
and perineum somatic nerves. Calcitonin gene-
related peptide (CGRP) and substance P (SP) in
dual labeled cells were determined by immunohistochemistry, giving
possible insight into the cause of pelvic pain
.Results: Fluorescent double labeled cells were found in the lumbar and
sacral DRG, while double labeled cells were distributed predominantly in
L6 to S1 and L1 to L2 segment DRG in groups 1 and 2, respectively. On
immunohistochemistry some of them were confirmed to contain CGRP
and SP. Thus, there are crossover pathways between the prostate and
pelvic floor.Conclusions: The findings that we present confirm that the
peripheral process of DRG cells dichotomizes to the prostate, sphincter
and somatic parties simultaneously. Some of these cells contain
CGRP and SP, which indicate that referred pain in the perineum and
pelvic floor may be caused by an axon reflex in the peripheral process of
DRG neurons.

L10 ANSWER 9 OF 12 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 4
AN 2004:398362 BIOSIS
DN PREV200400399374
TI Innervation of ectopic endometrium in a rat model of endometriosis.

AU Berkley, Karen J. [Reprint Author]; Dmitrieva, Natalia; Curtis, Kathleen
S.; Papka, Raymond E.
CS Program Neurosci, Florida State Univ, Tallahassee, FL, 32306, USA
kberkley@psy.fsu.edu
SO Proceedings of the National Academy of Sciences of the United States of
America, (July 27 2004) Vol. 101, No. 30, pp. 11094-11098. print.
ISSN: 0027-8424 (ISSN print).
DT Article
LA English
ED Entered STN: 13 Oct 2004
Last Updated on STN: 13 Oct 2004
AB Endometriosis (ENDO) is a disorder in which vascularized growths of
endometrial tissue occur outside the uterus. Its symptoms include reduced
fertility and severe pelvic pain. Mechanisms that
maintain the ectopic growths and evoke symptoms are poorly understood.
One factor not yet considered is that the ectopic growths develop their
own innervation. Here, we tested the hypothesis that the growths develop
both an autonomic and a sensory innervation. We used a rat model of
surgically induced ENDO whose growths mimic those in women. Furthermore,
similar to women with ENDO, such rats exhibit reduced fertility and
increased pelvic nociception. The ENDO was induced by autotransplanting,
on mesenteric cascade arteries, small pieces of uterus that formed
vascularized cysts. The cysts and healthy uterus were harvested from
proestrous rats and immunostained using the pan-neuronal marker PGP9.5 and
specific markers for calcitonin gene-related
peptide (CGRP) (sensory C and AS fibers), substance P
(SIP) (sensory C and AS fibers) and vesicular monoamine transporter
(sympathetic fibers). Cysts (like the uterus) were robustly innervated,
with many PGP9.5-stained neurites accompanying blood vessels and extending
into nearby luminal epithelial layers. CGRP-, SP-, and
vesicular monoamine transporter-immunostained neurites also were observed,
with CGRP and SP neurites extending the furthest into the cyst
lining. These results demonstrate that ectopic endometrial growths

develop an autonomic and sensory innervation. This innervation could contribute not only to symptoms associated with ENDO but also to maintenance of the ectopic growths.

L10 ANSWER 10 OF 12 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2004355964 EMBASE
TI Mechanisms in prostatitis/chronic pelvic pain syndrome.
AU Pontari, Michel A. (correspondence); Ruggieri, Michael R.
CS Department of Urology, Temple University School of Medicine, Philadelphia, PA, United States. Pontarm@tuhs.temple.edu
AU Pontari, Michel A. (correspondence)
CS Department of Urology, Temple University, Parkinson Pavilion, 3401 North Broad St., Philadelphia, PA 19140, United States. Pontarm@tuhs.temple.edu
SO Journal of Urology, (Sep 2004) Vol. 172, No. 3, pp. 839-845. Refs: 75
ISSN: 0022-5347 CODEN: JOURAA
CY United States
DT Journal; General Review; (Review)
FS 026 Immunology, Serology and Transplantation
028 Urology and Nephrology
003 Endocrinology
008 Neurology and Neurosurgery
LA English
SL English
ED Entered STN: 16 Sep 2004
Last Updated on STN: 16 Sep 2004
AB Purpose: We reviewed the current literature on mechanisms involved in the pathogenesis of prostatitis/chronic pelvic pain syndrome (CPPS). Materials and Methods: A literature review for the years 1966 to 2003 was performed using the MEDLINE database of the United States National Library of Medicine. Results: National Institutes of Health categories I and II prostatitis result from identifiable prostatic infections, whereas patients with category IV are asymptomatic. The majority of symptomatic cases are category III or chronic prostatitis (CP)/CPPS. The etiology of CP/CPPS is unknown. The traditional marker of inflammation, namely white blood cells in prostatic fluids, does not

correlate with the predominant symptom of pelvic pain. An imbalance toward increased proinflammatory and decreased anti-inflammatory cytokines has been implicated and a few studies have shown some correlation of this with pelvic pain. The imbalance in some men may result from polymorphisms at the cytokine loci. An autoimmune process may be involved and experimental evidence indicates that this can be under hormonal influence. Recent findings include possible defects in the androgen receptor. The prostate may not even be the source of the symptoms. Pelvic pain also correlates with the neurotrophin nerve growth factor implicated in neurogenic inflammation and central sensitization. Finally, psychological stress may produce measurable biochemical changes and influence the other processes. The role of normal prostatic bacterial flora in inciting the inflammatory response has also been reconsidered. Conclusions: The symptoms of CP/CPPS appear to result from an interplay between psychological factors and dysfunction in the immune, neurological and endocrine systems.

L10 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2002:256747 CAPLUS
DN 136:257266
TI Methods of diagnosing and treating small intestinal bacterial overgrowth and related conditions
IN Lin, Henry C.; Pimentel, Mark
PA USA
SO U.S. Pat. Appl. Publ., 53 pp., Cont.-in-part of U. S. Ser. No. 374,142.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 6

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20020039599	A1	20020404	US 2001-837797	20010417
US 7048906	B2	20060523		
CA 2220451	A1	19961121	CA 1996-2220451	19960516

US 5977175 A 19991102 US 1997-832307
19970403
US 6562629 B1 20030513 US 1999-374143
19990811
US 6861053 B1 20050301 US 1999-374142
19990811
US 20020094346 A1 20020718 US 1999-420046
19991018
US 6558708 B1 20030506 US 2000-546119
20000410
WO 2001011334 A2 20010215 WO 2000-US22168
20000811
WO 2001011334 A3 20010712
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA,
CH, CN,
CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH,
GM, HR,
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT,
RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ,
VN, YU,
ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE,
CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE,
BF, BJ,
CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
EP 1811303 A2 20070725 EP 2007-75358
20000811
EP 1811303 A3 20070815
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI,
LU, MC,
NL, PT, SE
CA 2444548 A1 20021024 CA 2002-2444548
20020416
WO 2002083926 A2 20021024 WO 2002-US12034
20020416
WO 2002083926 A3 20030515
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA,
CH, CN,
CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD,
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OM, PH,
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR,
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UA, UG, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT,
BE, CH,

CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002256254 A1 20021028 AU 2002-256254 20020416

AU 2002256254 B2 20070118

EP 1385476 A2 20040204 EP 2002-725704 20020416

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

JP 2005509588 T 20050414 JP 2002-582263 20020416

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20090828
PRAI US 1995-442843 B1 19950517
US 1997-832307 A1 19970403
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US 1999-374142 A2 19990811
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EP 2000-952739 A3 20000811
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AU 2001-251396 A3 20010407
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US 2001-837797 A 20010417
US 2002-107240 A3 20020326
AU 2002-256254 A3 20020416
JP 2002-582263 A3 20020416
WO 2002-US12034 W 20020416
US 2004-810020 A1 20040326
US 2004-853824 A3 20040526
US 2004-915193 A1 20040810
US 2005-234516 A3 20050923
US 2006-457445 A1 20060713
US 2007-838672 A1 20070814
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB Disclosed is a method of treating small intestinal bacterial overgrowth (SIBO) or a SIBO-caused condition in a human subject. SIBO-caused conditions include irritable bowel syndrome, fibromyalgia, chronic pelvic pain syndrome, chronic fatigue syndrome, depression, impaired mentation, impaired memory, halitosis, tinnitus, sugar craving, autism, attention deficit/hyperactivity disorder, drug sensitivity, an autoimmune disease, and Crohn's disease. Examples are provided showing effects of antibiotics on SIBO, demonstrating the roles of peptide YY and the serotoninergic/adrenergic/opioid pathways in SIBO, and the effects of ondansetron, propranolol, norepinephrine and naloxone on intestinal transit. The invention thus relates to slowing upper gastrointestinal transit, thereby enhancing the digestion and/or absorption of predigested nutrients. Gastrointestinal transit-slowing compns. comprise active agents such as lipids, serotonin, serotonin

agonists, serotonin re-uptake inhibitors, peptide YY, calcitonin gene-related peptide, adrenergic agonists and opioid agonists. Also disclosed are a method of screening for the abnormally likely presence of SIBO in a human subject and a method of detecting SIBO in a human subject. A method of determining the relative severity of SIBO or a SIBO-caused condition in a human subject, in whom small intestinal bacterial overgrowth has been detected, is also disclosed.

OSC.G 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

RE.CNT 251 THERE ARE 251 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 12 OF 12 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 5

AN 2001:24473 BIOSIS

DN PREV200100024473

TI Growth of nerve fibres into murine peritoneal adhesions.

AU Sulaiman, Hassan; Gabella, Giorgio; Davis, Christine; Mutsaers, Steven E.; Boulos, Paul; Laurent, Geoffrey J.; Herrick, Sarah E. [Reprint author]

CS Department of Medicine, Rayne Institute, University College London, 5 University Street, London, WC1E 6JJ, UK
s.herrick@ucl.ac.uk

SO Journal of Pathology, (November, 2000) Vol. 192, No. 3, pp. 396-403.
print.
CODEN: JPTLAS. ISSN: 0022-3417.

DT Article

LA English

ED Entered STN: 3 Jan 2001
Last Updated on STN: 12 Feb 2002

AB Adhesions in the peritoneal cavity have been implicated in the cause of intestinal obstruction and infertility, but their role in the aetiology of chronic pelvic pain is unclear. Nerves have been demonstrated in human pelvic adhesions, but the presence of pain-conducting fibres has not been established. The purpose of this study was to use an animal model to examine the growth of nerves during adhesion formation at various times following injury and to characterize the types of fibres present. Adhesions were generated in mice by injuring

the surface of the caecum and adjacent abdominal wall, with apposition.
At 1-8 weeks post-surgery, adhesions were processed and nerve fibres
characterized histologically, immunohistochemically, and ultrastructurally. Peritoneal adhesions had consistently formed by 1 week
after surgery and from 2 weeks onwards, all adhesions contained some nerve
fibres which were synaptophysin, calcitonin gene-related peptide, and substance P-immunoreactive, and were seen to originate from the caecum. By 4 weeks post-surgery, nerve
fibres were found to originate from both the caecum and the abdominal
wall, and as demonstrated by acetylcholinesterase histochemistry, many
traversed the entire adhesion. Ultrastructural analysis showed both
myelinated and non-myelinated nerve fibres within the adhesion. This
study provides the first direct evidence for the growth of sensory nerve
fibres within abdominal visceral adhesions in a murine model and suggests
that there may be nerve fibres involved in the conduction of pain stimuli.

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FULL ESTIMATED COST	0.07	235.17

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	

-2.55

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